Mortality Model Based on Delays in Progression of Chronic Diseases: Alternative to Cause Elimination Model

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BILLIONS HAVE BEEN SPENT ON RESEARCH and treatment to eliminate or significantly reduce the major chronic disease killers of the U.S. population. Frequently, life tables based on cause elimination are used to assess the health impact of reducing these causes of death. The results of such analyses, however, have often been criticized as being unrealistic. One criticism is that death is often not due to a single disease, but to many causes, and that diseases may operate interdependently. Recent articles have focused upon the analysis of multiple cause of death data and the ways in which disease interdependence might be modeled (*1*). A second criticism of the cause elimination models is that the total elimination of a major cause of death is not biomedically feasible.

There are two basic concepts of how causes can be eliminated. The first is by prevention. Typically, such efforts would eliminate some proportion of the hazard associated with a specific cause of death. For example, it has been suggested recently that 80 percent of cancer is attributable to controllable environmental influences. Presumably, then, control of environmental hazards could prevent up to 80 percent of cancer mortality by preventing up to 80 percent of the disease's incidence. Prevention can thus be equated with cause elimination since the disease never occurs. The impact on health of the complete elimination of a cause of death can be assessed by the cause elimination life table model. If, however, prevention is restricted to some proportion of the mortality due to a given cause, the expected change in life expectancy can be assessed by the methods proposed by Tsai and associates (*2*).

An alternate rationalization of cause elimination is based on the delay of the age at death due to a specific disease. This delay could be accomplished in one of several ways. First, improvement in treatment may slow the progress of the disease. Second, certain associated causes of death that interact with the underlying condition to hasten death may be eliminated. This latter type of delay modeling would be consistent with multiple cause analysis when an underlying cause of death can be identified and mortality is reduced by eliminating the associated causes of death. In either case, the underlying disease continues to exist, but the afflicted person survives much longer with it. The third way a delay might be achieved is by slowing the age

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changes that lead to the disease. In this case, the onset of the disease is delayed. In all cases we are dealing with the delay of a person's death due to a specific cause and the likelihood that the person will not experience the full delay possible because of the competing risk of other causes of death.

For a variety of reasons, an approach to mortality reduction based on delay of death is perhaps a more reasonable representation of the impact that biomedical advances will have in the near future in the United States on the mortality hazards associated with the chronic degenerative diseases. For example, life expectancy might be increased by 3 to 5 years simply by changes in lifestyle—the elimination of cigarette smoking and changes in nutrition (*3*). However, even when some factor, such as smoking, can be clearly identified with excess mortality risk, inducing people to change their personal habits is difficult. Similarly, environmental controls are hard to impose on a broad basis because of concern about their economic consequences and because of the political influence of corporate interests. Practically speaking, the likely changes in lifestyle and in the control of environmental hazards may not be sufficient to prevent an increase in the associated mortality hazards. Consequently, major improvements in public health may have to come from innovations in the medical management of chronic diseases. People who are thereby saved, however, will still manifest the chronic disease, and hence the prevalence of chronic diseases in the population will increase, along with the associated costs of treatment and care.

A model of mortality reductions achieved by delaying death avoids certain logical contradictions that are manifest in the cause elimination life table model. Specifically, everyone will eventually die of some disease. Assessment of the impact that the individual elimination of each of the major causes of death would have on survival suggests that elimination of all of these causes of death is hypothetically possible, but the researcher is then left with the question of what kills the person. Under the delayed mortality model, if the major causes of death are "delayed" by 3 years, the gain in average life expectancy will be approximately 3 years, even though the proportion of all persons dying from each of the major causes of death will remain constant. Thus, the logical problem of a "deathless" population will not arise.

We present the details of a method for determining the effect of a given delay in death from a selected cause in the presence of other competing causes of death, as well as examples of what the estimated effects would have been on the U.S. population in 1969 of delays of 5, 10, and 15 years in the ages at death from three major chronic degenerative diseases.

Methods

The calculations leading to cause elimination life tables are often based on the theory of competing risks (*4*). In this theory, each of the r potential causes of death (risks) is considered to add an independent increment to the force of mortality. Thus, the total force of mortality is simply the sum of the component forces. Additionally, it is assumed that associated with each risk is a distribution of "times to die." Since a person can only die once, it follows that the risk, d, with the smallest time to die, t_d, will be the cause of death for that person. Under the assumption of independent risks, the r times to die will also be independent (conditional on age). Because a person dies from the cause d that has the minimum time t_d, the causes are said to be competing for the life of the person. Elimination of cause d is equivalent to having an infinite t_d, since in this case t_d can never be the minimum of t; thus, the implication is that risk d will never be a cause of death. It follows that death will be delayed by an amount equal to $t_c - t_d$ where t_c is the minimum of the remaining $(r - 1)$ times to die. In this case, the "saved" person is returned to the risks of the population unaffected by d.

In this paper, we are concerned with the effects of delaying the times to die from various causes rather than the effects of eliminating these causes. Delaying cause d by some number of years, say l_d, is equivalent to replacing t_d by $t_d + l_d$. In this case, the saved person may still die of d if $l_d < t_c - t_d$. However, if $l_d > t_c - t_d$, then the saved person will die of c. Thus a delay of l_d years will have the effect of shifting some proportion of deaths due to d to other causes. In contrast, the cause elimination life table model for cause d shifts all deaths due to d to other causes.

We now consider how one calculates cause-adjusted life tables based upon the cause-delay concept. First, since the risks are assumed to be independent, we need to consider only two risks—d, the disease of interest, and c, which represents all other causes of death. Second, if we assume that the force of mortality due to d is a constant proportion of the total force of mortality over the i^{th} age interval (x_i, x_{i+1}), it follows that the cumulative (over the i^{th} age interval) forces of mortality due to d, F_{id}, and c, F_{ic}, are given by

$$F_{id} = F_i \, q_{id} \div q_i \qquad \text{(equation 1a)}$$

and

$$F_{ic} = F_i \, q_{ic} \div q_i, \qquad \text{(equation 1b)}$$

where

$$F_i = -\ln(1 - q_i) \qquad \text{(equation 2)}$$

is the cumulative total force of mortality and where ln denotes the natural logarithm. In equation 1, q_{id} is the crude probability of death due to d, q_{ic} is the crude probability of death due to c, and q_i is the crude probability of death due to all causes. Thus,

$$q_i = q_{id} + q_{ic}.$$

Also, from equations 1 and 2 we find that

$$q_i = 1 - \exp(-F_i) \quad \text{(equation 3)}$$

where $F_i = F_{id} + F_{ic}$.

Now, since t_d is a random variable, it follows that delaying d by l_d years has the effect of delaying the time at which a given force of mortality will be operative by l_d years. Thus, if we can assume that l_d is a multiple of the age interval size n (where $n = x_{i+1} - x_i$, for all i), it follows that

$$F_{id}(l_d) = F_{(i-m)d}, \quad x_i \geqq l_d \quad \text{(equation 4)}$$
$$= 0, \quad x_i < l_d$$

where $m = l_d \div n$.

In this case, the adjusted cumulative total force of mortality, $F_i(l_d)$, is given by

$$F_i(l_d) = F_{id}(l_d) + F_{ic}, \quad \text{(equation 5)}$$

and the adjusted crude probability of death due to all causes, $q_i(l_d)$, is given by

$$q_i(l_d) = 1 - \exp\{-F_i(l_d)\}. \quad \text{(equation 6a)}$$

The adjusted life table can be generated by using standard calculations based on the $q_i(l_d)$ derived from equation 6a.

Equation 6a shows the adjusted probability, $q_i(l_d)$, as a function of the adjusted cumulative total force of mortality, $F_i(l_d)$. Since published life tables do not have an F_x column, we will express $q_i(l_d)$ as a function of the crude probabilities. Substitution of equation 5 in equation 6a and simplification yields

$$q_i(l_d) = 1 - (1 - q_{i-m})^{r_{(i-m)d}} (1 - q_i)^{r_{ic}} \quad \text{(equation 6b)}$$

where

$$r_{(i-m)d} = q_{(i-m)d} \div q_{i-m}$$
$$r_{ic} = q_{ic} \div q_i.$$

Note that for $m = 0$, equation 6b is the identity

$$q_i(l_d = 0) = q_i.$$

For $m > i$, equation 6b becomes

$$q_i(l_d) = 1 - (1 - q_i)^{q_{ic} \div q_i},$$

which, according to Chiang (*4*), is the net probability, $q_{i \cdot d}$, that a person alive at age x_i would die before x_{i+1} if d were eliminated as a cause of death. Thus, cause elimination is equivalent to an infinite delay, that is $l_d = \infty$.

The effects of a given delay of a cause of death can be measured as the difference, $\triangle \mathring{e}_i(l_d)$, in life expectancy before and after the delay, that is,

$$\triangle \mathring{e}_i(l_d) = \mathring{e}_i(l_d) - \mathring{e}_i. \quad \text{(equation 7)}$$

In equation 7, $\mathring{e}_i$ is derived from life table calculations based on the crude probabilities q_i, and $\mathring{e}_i(l_d)$ is derived by using the adjusted crude probabilities $q_i(l_d)$. In comparing the effects of various delays, l_d's, relative to the effects of cause elimination, we would use the ratio $P_i(l_d)$, where

$$P_i(l_d) = \triangle \mathring{e}_i(l_d) \div \triangle \mathring{e}_i(\infty). \quad \text{(equation 8)}$$

From equation 8 it is seen that $P_i(l_d)$ is the ratio of the gain in life expectancy due to the delay of l_d years to the gain under total elimination of d. Thus, $P_i(l_d)$ represents the proportion of the maximum possible gain that is associated with the given delay.

Results

The life expectancies that would occur if a given disease were to be delayed for a specified number of years are determined by a simple modification of standard life table techniques. Specifically, the observed crude probabilities q_i are replaced by the adjusted probabilities $q_i(l_d)$, which are derived from the crude probabilities q_i and q_{id}. With this substitution, the standard life table computations may be applied to generate the adjusted age-specific life expectancies. Using Chiang's method (*4*), we thus derived life expectancy figures based on complete current life table calculations for white and black males and females in the United States in 1969. These calculations were performed independently for three major causes of death representing chronic degenerative diseases—cancer, ischemic heart disease, and stroke—at five hypothetical levels of delay —0, 5, 10, and 15 years, and for an infinite delay (∞).

We derived the data for these calculations from two sources. First, age-race-sex-cause-specific mortality counts, D_{id}, were tabulated from microdata mortality tapes provided by the National Center for Health Statistics. These tapes contained codings from the International Classification of Diseases, Adapted, 8th Revision, of the underlying cause for each death occurring in the United States in 1969. Each underlying cause was then classified into one of the three disease categories of interest or to a residual category. Thus, for any given

disease, d, we defined c to include the residual category plus the two other diseases. Second, age-race-sex population data, N_i's, were derived by backdating the adjusted census data of Siegel (*5*) to January 1, 1969. Combining these two data sources yielded the required crude probabilities for use in equation 6b:

$$q_{id} = D_{id} \div N_i; \qquad \text{(equation 9)}$$

$$q_{ic} = D_{ic} \div N_i;$$

$$\text{and } q_i = q_{id} + q_{ic}.$$

In table 1, the life expectancies in years at selected ages for the four race-sex groups for various delays of cancer are shown. Note that the 0-year delay corresponds to the observed life expectancies; the infinite delay corresponds to the life expectancies determined by the standard cause elimination methods. For example, the table shows that at birth (age zero) white males have an observed life expectancy (zero years delay column) of 68.0 years. If cancer were completely eliminated as a cause of death (the infinite delay column), then white males would have their life expectancy at birth increased to 70.3 years—an increase of 2.3 years. At age 65, white males have an observed life expectancy of 13.1 years, a figure indicating that persons in this category will, on the average, live to age 78.1 (that is, 65.0 + 13.1 years). If cancer were eliminated, however, the age 65 life expectancy would be increased to 14.6 years—an increase of 1.5 years. The gains in life expectancy to be realized if cancer were eliminated as a cause of death can be computed for other ages and demographic groups as the differences between the infinite delay columns and the zero year delay columns, as indicated in equation 7.

If cancer mortality is delayed by 10 years, then we see from table 1 that white male life expectancy at birth would be increased to 69.3 years—an increase of 1.3 years. This increase may be compared with the 2.3-year increase associated with the complete elimination of cancer mortality noted in the preceding paragraph. Using equation 8 we find that the 1.3-year gain associated with a 10-year delay of cancer mortality represents 57 percent ($1.3 \div 2.3 \times 100$) of the 2.3-year maximum gain.

The 10-year delay in cancer mortality would increase the white male life expectancy at age 65 from 13.1 to 13.7 years—an increase of 0.6 years, a figure representing 40 percent of the 1.5-year gain to be realized at age 65 if cancer were completely eliminated.

Calculations based on data in table 1 show that at birth the percent gain in life expectancy from delaying cancer is similar for white males and black males (35 percent versus 30 for a 5-year delay, 57 percent versus 52 for a 10-year delay, and 74 percent versus 70 for a 15-year delay). White females and black females show a relatively smaller proportionate gain in life expectancy at birth (white females—27 percent for a 5-year delay, 46 percent for a 10-year delay, and 65 percent for a 15-year delay and black females—24 percent, 44 percent, and 56 percent for the corresponding delays). With increasing age, the relative differences in the effects of delay decrease.

Table 2 shows the effects on death of retardation of the progress of ischemic heart disease (IHD). For this disease, the percentage of the total possible gain in life expectancy with a 5-, 10-, and 15-year delay in death is roughly the same for white males (31, 53, and 69 percent) and black males (27, 49, and 64 percent). Black females show the smallest proportionate gain for the three hypothetical delays (25, 46, and 61 percent). Interestingly, for white males and both black groups, the percentage gains at birth from delays in cancer deaths (table 1) and from delays in IHD deaths (table 2) are remarkably similar. White females, on the other hand, show a much greater percentage increase in life expectancy from delays in IHD deaths (34, 60, and 77 percent for 5-, 10-, and 15-year delays). Thus, for a 15-year delay in IHD deaths, the percentage gain in life expectancy at birth for white females has increased by 12 percent over that for the delay of cancer deaths. In contrast to cancer, the proportion of the gain in life expectancy due to a specific delay in IHD deaths is approximately maintained to advanced ages. The reason is that IHD, unlike cancer, does not exhibit a downturn in mortality at advanced ages.

Table 3 shows the effects of delaying stroke deaths for varying periods. We see that both white groups have almost the same percentage gain in life expectancy for a 5-, 10-, and 15-year delay (44, 67, and 89 for white males; 43, 71, and 86 for white females). Both black groups, on the other hand show a much smaller percentage change with an increased delay (33, 53, and 73 for black males and 29, 50, and 67 for black females). Examination of the percentage gains at other ages shows that the smaller percentage gain for a given delay for blacks is maintained to advanced ages.

Discussion

The preceding results show the effects on survival of delaying death from cancer, ischemic heart disease, and stroke for specified periods. They clearly show that even a moderate retardation—say 5 years—in the rate of progression of a chronic disease will yield a sizable portion of the total change in longevity that might be

Table 1. Calculated effects on life expectancy at selected ages from delaying

At age	White males' life expectancy with delay of—					White females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	68.0	68.8	69.3	69.7	70.3	75.6	76.3	76.8	77.3	78.2
5	64.7	65.4	66.0	66.3	67.0	72.0	72.7	73.3	73.7	74.6
10	59.9	60.6	61.1	61.5	62.2	67.2	67.8	68.4	68.8	69.7
15	55.0	55.7	56.2	56.6	57.3	62.2	62.9	63.5	63.9	64.8
20	50.4	51.1	51.6	52.0	52.7	57.4	58.1	58.6	59.0	60.0
25	45.9	46.6	47.1	47.5	48.1	52.6	53.3	53.8	54.2	55.1
30	41.2	41.9	42.5	42.8	43.5	47.8	48.5	49.0	49.4	50.3
35	36.6	37.3	37.8	38.2	38.8	43.0	43.6	44.2	44.6	45.5
40	32.0	32.7	33.2	33.6	34.2	38.3	38.9	39.4	39.8	40.7
45	27.6	28.3	28.8	29.1	29.8	33.7	34.3	34.7	35.1	36.0
50	23.4	24.1	24.5	24.9	25.5	29.3	29.8	30.2	30.5	31.4
55	19.6	20.1	20.6	20.9	21.5	25.0	25.4	25.8	26.1	26.9
60	16.2	16.6	17.0	17.3	17.9	21.0	21.3	21.5	21.8	22.5
65	13.1	13.4	13.7	14.0	14.6	17.1	17.3	17.6	17.7	18.4
70	10.5	10.7	10.9	11.1	11.6	13.6	13.8	13.9	14.1	14.5
75	8.2	8.4	8.5	8.7	9.1	10.5	10.6	10.7	10.8	11.2
80	6.3	6.4	6.5	6.6	6.9	7.8	7.8	8.0	8.0	8.3
85	4.9	4.9	4.9	5.0	5.3	5.7	5.8	5.8	5.9	6.0
90	3.7	3.7	3.8	3.8	4.0	4.3	4.3	4.3	4.3	4.5
95	3.2	3.2	3.2	3.2	3.3	3.6	3.6	3.6	3.6	3.8

NOTE: The 0-year delay corresponds to the observed life expectancies; the infinite delay corresponds to the life expectancies determined by standard

gained from the total elimination of the disease. The three diseases selected for study are all chronic degenerative diseases that possibly correlate with intrinsic aging processes and that have recently shown decreases in mortality. To model these mortality reductions as a delay in pathological processes, rather than as the elimination of a portion of disease incidence, seems more realistic.

Specifically, although as high as 80 percent of cancers may be environmentally determined, the rapid elimination of those cancers that are not genetically caused may not be feasible because of the potential economic disruption from tighter environmental standards and the difficulty in modifying lifestyles (for example, by the elimination of cigarette smoking). Indeed, the results of efforts at the environmental control of suspected carcinogens and at getting people to change their lifestyles suggest that even keeping cancer incidence as low as it is will be difficult. In addition, since cancer incidence is strongly related to age, basic aging processes may play a considerable role in its onset, and those age changes that lead to cancer's onset would be difficult to eliminate. Nevertheless, retardation of these changes might become biomedically feasible, although in those people who did not die earlier of some other disease cancer would still develop, but at a later age.

Alternately, changes in cancer mortality might be realized by increasing the survival time of cancer patients. Indeed, increasing survival time has been the national trend recently. The effects of increasing cancer survival by 5, 10, and 15 years are illustrated in table 1.

The bulk of ischemic heart disease and stroke, on the other hand, may be related to the process of atherogenesis. Atherogenesis seems to be a condition manifested to some degree in most populations past the age of 30. Diet, exercise, and new chemotherapeutic agents may contribute to slowing this process, but its elimination is unlikely. Effective medical management of the acute phase of ischemic heart disease may also contribute to the delay of death by helping a person survive what, without an effective response, would be a lethal cardiovascular episode. This achievement in no way, however, implies control of the underlying disease process.

For stroke, the likelihood of increasing the survival of acute disease episodes may be small. However, the retardation of atherogenesis, as well as the control of hypertension, an associated morbid condition that can trigger early circulatory catastrophe, might significantly delay stroke deaths. Thus, it is more realistic to model reductions in the mortality from each of these

cancer in 1969 U.S. population for 5, 10, and 15 years and infinity

At age	Black males' life expectancy with delay of—					Black females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	61.2	61.9	62.4	62.8	63.5	69.1	69.7	70.2	70.5	71.6
5	59.0	59.7	60.3	60.6	61.4	66.6	67.2	67.7	68.1	69.2
10	54.2	54.9	55.4	55.8	56.6	61.8	62.4	62.9	63.3	64.3
15	49.4	50.1	50.6	51.0	51.8	56.9	57.5	58.0	58.4	59.5
20	44.9	45.6	46.2	46.6	47.4	52.2	52.7	53.2	53.6	54.7
25	40.7	41.4	42.0	42.4	43.2	47.5	48.1	48.6	49.0	50.0
30	36.5	37.2	37.8	38.2	39.0	42.9	43.5	44.0	44.4	45.4
35	32.3	33.1	33.6	34.0	34.8	38.4	39.0	39.5	39.9	40.9
40	28.3	29.1	29.6	30.0	30.8	34.2	34.7	35.2	35.5	36.6
45	24.6	25.3	25.8	26.2	27.1	30.2	30.6	31.1	31.4	32.5
50	21.1	21.6	22.2	22.6	23.4	26.4	26.7	27.1	27.4	28.5
55	17.9	18.4	18.9	19.3	20.1	22.9	23.1	23.4	23.7	24.7
60	15.0	15.4	15.8	16.1	17.0	19.5	19.7	19.9	20.1	21.1
65	12.5	12.7	13.0	13.3	14.2	16.6	16.7	16.9	17.1	17.9
70	10.7	10.8	11.0	11.3	12.1	14.6	14.6	14.7	14.8	15.6
75	9.2	9.3	9.4	9.6	10.4	12.6	12.6	12.6	12.7	13.3
80	7.8	7.8	7.8	7.9	8.7	10.4	10.4	10.4	10.4	10.9
85	6.9	6.9	6.9	7.0	7.6	8.7	8.7	8.7	8.7	9.1
90	6.3	6.3	6.3	6.3	6.9	7.1	7.1	7.1	7.1	7.4
95	5.6	5.7	5.6	5.6	6.1	5.9	5.9	5.9	5.9	6.1

cause elimination methods.

diseases as delays in the age at death rather than as the complete elimination of the disease in individuals.

The mortality delay model also provides a new basis for predicting future changes in mortality patterns. For example, because certain chronic diseases are related to basic aging changes, elimination of all the mortality associated with them is unlikely. However, estimating what percentage of the mortality for a given chronic disease can be eliminated is worth considering. Provision of an estimated percentage does not alter the fact that some proportion of the population will still die from the same chronic disease, but those deaths will occur at an older age. The mortality delay model can serve as a basis for estimating from empirical sources the amount of the delay in death.

For example, data on increases in the median survival time of cancer patients undergoing new types of treatment at major medical centers are available. Presumably, these patients represent the optimal level of survival for patients with this disease, given the existing state of medical technology. By using the increase in median survival time so derived to estimate probable delays in mortality, we can, in effect, produce life tables that would represent the survival patterns of cancer patients in the U.S. population provided that the best available medical care were disseminated to the entire population.

A similar empirical procedure can be used to determine delays in death when death has multiple causes. Specifically, people who have only cancer may die at later ages than people with both cancer and pneumonia. The difference between the ages of these two groups at death would represent the period of delay in the onset of death that is appropriate for people whose cancer was an underlying cause of death but whose pneumonia had been effectively treated. Such analysis, however, would have to be carefully conducted, since the occurrence of certain complications may correlate with aging. Thus, if only elderly people died with pneumonia as a complication, the results could be anomalous unless one were careful to look at age-specific delays in mortality (that is, mortality delays for a specific disease that varied systematically with age).

Clearly, the mortality delay model provides a framework for arriving at empirically based estimates of mortality reduction patterns. And in planning public health and social programs that depend upon realistic projections of a population's or population group's survivorship, such as that of the elderly, knowledge of these patterns is extremely valuable (*6*).

Table 2. Calculated effects on life expectancy at selected ages from delaying

At age	White males' life expectancy with delay of—					White females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	68.0	69.8	71.1	72.0	73.8	75.6	77.2	78.4	79.2	80.3
5	64.7	66.5	67.8	68.8	70.6	72.0	73.7	74.9	75.7	76.8
10	59.9	61.6	63.0	64.0	65.7	67.2	68.8	70.0	70.8	71.9
15	55.0	56.8	58.1	59.1	60.9	62.2	63.9	65.1	65.9	67.0
20	50.4	52.2	53.6	54.5	56.3	57.4	59.1	60.3	61.1	62.2
25	45.9	47.7	49.0	50.0	51.8	52.6	54.3	55.5	56.3	57.4
30	41.2	43.0	44.4	45.4	47.2	47.8	49.5	50.6	51.4	52.6
35	36.6	38.4	39.8	40.8	42.6	43.0	44.7	45.9	46.7	47.8
40	32.0	33.8	35.2	36.2	38.0	38.3	40.0	41.2	42.0	43.1
45	27.6	29.3	30.7	31.7	33.5	33.7	35.4	36.6	37.4	38.6
50	23.4	25.0	26.3	27.3	29.2	29.3	31.0	32.2	33.0	34.1
55	19.6	21.1	22.3	23.2	25.1	25.0	26.7	27.9	28.7	29.9
60	16.2	17.5	18.5	19.4	21.3	21.0	22.6	23.7	24.5	25.7
65	13.1	14.2	15.2	16.0	17.8	17.1	18.6	19.8	20.5	21.7
70	10.5	11.5	12.3	13.0	14.7	13.6	15.0	16.0	16.8	18.0
75	8.2	9.1	9.8	10.4	11.9	10.5	11.7	12.6	13.3	14.5
80	6.3	7.1	7.7	8.2	9.6	7.8	8.8	9.7	10.3	11.4
85	4.9	5.4	6.0	6.4	7.7	5.7	6.5	7.2	7.8	8.9
90	3.7	4.2	4.6	5.0	6.2	4.3	4.9	5.4	5.9	7.0
95	3.2	3.4	3.8	4.1	5.3	3.6	3.9	4.3	4.8	5.9

NOTE: The 0-year delay corresponds to the observed life expectancies; the Infinite delay corresponds to the life expectancies determined by standard

Table 3. Calculated effects on life expectancy at selected ages from delaying

At age	White males' life expectancy with delay of—					White females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	68.0	68.4	68.6	68.8	68.9	75.6	76.2	76.6	76.8	77.0
5	64.7	65.1	65.3	65.5	65.6	72.0	72.6	73.0	73.2	73.5
10	59.9	60.2	60.5	60.6	60.8	67.2	67.8	68.1	68.3	68.6
15	55.0	55.4	55.6	55.7	55.9	62.2	62.9	63.2	63.4	63.7
20	50.4	50.8	51.0	51.2	51.3	57.4	58.0	58.4	58.6	58.9
25	45.9	46.3	46.5	46.6	46.8	52.6	53.2	53.6	53.8	54.1
30	41.2	41.6	41.8	42.0	42.1	47.8	48.4	48.8	49.0	49.2
35	36.6	37.0	37.2	37.3	37.5	43.0	43.6	44.0	44.2	44.4
40	32.0	32.4	32.6	32.8	32.9	38.3	38.9	39.3	39.5	39.7
45	27.6	28.0	28.2	28.4	28.5	33.7	34.3	34.7	34.9	35.2
50	23.4	23.8	24.1	24.2	24.4	29.3	29.9	30.3	30.5	30.7
55	19.6	20.0	20.2	20.4	20.5	25.0	25.6	26.0	26.2	26.5
60	16.2	16.5	16.8	16.9	17.0	21.0	21.6	21.9	22.1	22.4
65	13.1	13.5	13.7	13.9	14.0	17.1	17.7	18.0	18.2	18.5
70	10.5	10.9	11.1	11.2	11.4	13.6	14.1	14.5	14.7	14.9
75	8.2	8.6	8.8	9.0	9.1	10.5	11.0	11.3	11.5	11.7
80	6.3	6.6	6.8	7.0	7.2	7.8	8.2	8.5	8.7	9.0
85	4.9	5.1	5.3	5.4	5.6	5.7	6.1	6.3	6.5	6.7
90	3.7	3.9	4.0	4.1	4.3	4.3	4.5	4.7	4.9	5.1
95	3.2	3.3	3.4	3.5	3.7	3.6	3.7	3.9	4.0	4.3

NOTE: The 0-year delay corresponds to the observed life expectancies; the Infinite delay corresponds to the life expectancies determined by standard

ischemic heart disease in 1969 U.S. population for 5, 10, and 15 years and infinity

At age	Black males' life expectancy with delay of—					Black females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	61.2	62.4	63.4	64.1	65.7	69.1	70.5	71.7	72.6	74.8
5	59.0	60.3	61.3	62.0	63.7	66.6	68.1	69.3	70.3	72.6
10	54.2	55.5	56.5	57.2	58.9	61.8	63.3	64.5	65.4	67.7
15	49.4	50.7	51.7	52.4	54.1	56.9	58.4	59.6	60.5	62.9
20	44.9	46.2	47.3	48.0	49.7	52.2	53.7	54.9	55.8	58.1
25	40.7	42.1	43.1	43.9	45.5	47.5	49.0	50.2	51.1	53.5
30	36.5	37.8	38.9	39.7	41.4	42.9	44.4	45.6	46.6	48.9
35	32.3	33.7	34.7	35.5	37.3	38.4	40.0	41.2	42.1	44.6
40	28.3	29.6	30.7	31.5	33.3	34.2	35.7	36.9	37.9	40.4
45	24.6	25.8	26.9	27.7	29.5	30.2	31.7	32.9	33.9	36.4
50	21.1	22.2	23.2	24.0	25.9	26.4	27.8	29.0	30.0	32.6
55	17.9	19.0	19.9	20.7	22.6	22.9	24.2	25.4	26.3	29.0
60	15.0	16.0	16.8	17.6	19.6	19.5	20.8	21.9	22.8	25.5
65	12.5	13.3	14.1	14.8	16.8	16.6	17.7	18.8	19.7	22.4
70	10.7	11.3	12.1	12.7	14.8	14.6	15.4	16.4	17.3	20.1
75	9.2	9.8	10.3	10.9	13.0	12.6	13.4	14.1	14.9	17.7
80	7.8	8.3	8.8	9.2	11.3	10.4	11.3	11.9	12.4	15.2
85	6.9	7.2	7.7	8.1	10.8	8.7	9.4	10.2	10.7	13.3
90	6.3	6.5	6.8	7.2	9.2	7.1	7.8	8.4	8.9	11.4
95	5.6	5.8	6.1	6.3	8.2	5.9	6.4	7.0	7.4	10.0

cause elimination methods.

stroke in 1969 U.S. population for 5, 10, and 15 years and infinity

At age	Black males' life expectancy with delay of—					Black females' life expectancy with delay of—				
	0 years	5 years	10 years	15 years	∞ years	0 years	5 years	10 years	15 years	∞ years
0	61.2	61.7	62.0	62.3	62.7	69.1	69.8	70.3	70.7	71.5
5	59.0	59.5	59.9	60.1	60.6	66.6	67.4	67.9	68.3	69.2
10	54.2	54.7	55.1	55.3	55.8	61.8	62.5	63.1	63.5	64.3
15	49.4	49.9	50.2	50.5	50.9	56.9	57.6	58.2	58.6	59.4
20	44.9	45.4	45.8	46.0	46.5	52.2	52.9	53.4	53.8	54.7
25	40.7	41.2	41.6	41.9	42.3	47.5	48.2	48.8	49.2	50.0
30	36.5	37.0	37.4	37.6	38.1	42.9	43.6	44.2	44.6	45.4
35	32.3	32.8	33.2	33.5	33.9	38.4	39.2	39.7	40.1	41.0
40	28.3	28.8	29.2	29.5	30.0	34.2	34.9	35.4	35.9	36.7
45	24.6	25.1	25.4	25.7	26.2	30.2	30.9	31.4	31.8	32.7
50	21.1	21.5	21.9	22.1	22.6	26.4	27.1	27.6	28.0	28.9
55	17.9	18.4	18.7	19.0	19.5	22.9	23.5	24.0	24.4	25.3
60	15.0	15.4	15.8	16.0	16.5	19.5	20.1	20.6	21.0	21.9
65	12.5	12.9	13.2	13.4	14.0	16.6	17.2	17.7	18.0	18.9
70	10.7	11.0	11.3	11.6	12.1	14.6	15.0	15.5	15.8	16.7
75	9.2	9.5	9.7	10.0	10.6	12.6	12.9	13.3	13.6	14.5
80	7.8	8.0	8.2	8.4	9.0	10.4	10.7	11.0	11.2	12.1
85	6.9	7.1	7.3	7.4	8.1	8.7	9.0	9.3	9.4	10.2
90	6.3	6.3	6.5	6.6	7.3	7.1	7.4	7.6	7.8	8.4
95	5.6	5.7	5.7	5.8	6.4	5.9	6.1	6.3	6.4	7.0

cause elimination methods.

Although the gains in life expectancy to be achieved by delaying the rate of progression of a disease are less than the gain that would be achieved under the total elimination of the disease, the magnitude of those gains will still be important for policy purposes. For example, we previously indicated that a 10-year delay in cancer mortality would increase the white male life expectancy at age 65 by only 0.6 years, (that is, from 13.1 to 13.7 years). Since 65 is the age when social security benefits usually start, we see that the 0.6-year gain implies that each white male social security recipient would have a 5 percent (that is, $0.6 \div 13.1 \times 100$) increase in the period that he received benefits with little or no increase in monetary contributions to the system. If the value of these benefits remained constant over time, his expected total lifetime benefits would increase by 5 percent. Then, since this 0.6-year gain in life expectancy would apply to all white males at age 65, we would conclude that social security costs for white males would increase by 5 percent over current costs if cancer mortality were delayed 10 years.

Similar calculations may be derived for other demographic groups or disease categories. Also, depending on how the delay in death is actually accomplished (prevention versus treatment), there might be substantial additional costs that would have to be determined for input to policy analyses. Thus, the prevention or delay of deaths due to the major chronic diseases could significantly affect public policy.

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SYNOPSIS

MANTON, KENNETH G. (Duke University Center for Demographic Studies), PATRICK, CLIFFORD H., and STALLARD, ERIC: *Mortality model based on delays in progression of chronic diseases: alternative to cause elimination model. Public Health Reports, Vol. 95, November–December 1980, pp. 580–588.*

For the analysis of the impact of major chronic diseases on a population, a life table model is proposed in which the age at death due to a specific cause (chronic disease) is postponed. Even though many of the major causes of death related to intrinsic aging processes are impossible to eliminate, these causes might be significantly delayed or retarded. To illustrate the use of this model, the effects of a delay of 5, 10, and 15 years in deaths due to three chronic degenerative diseases (cancer, ischemic heart disease, and stroke) are calculated for specific race-sex components of the U.S. population in 1969. These calculations show that even moderate delays in the progression of major chronic diseases will yield a sizable portion of the total gain in longevity that would be available if the diseases were totally eliminated. Thus, they demonstrate that a life table model based on cause delay provides a more biomedically plausible representation of the health impact of a chronic disease on a population than does the cause elimination life table model. Additionally, the cause-delay model provides a mechanism for incorporating the likely effects of medical innovation on survival.